

ROLE OF PUMP PRIME IN THE ETIOLOGY OF CARDIO-PULMONARY BYPASS ASSOCIATED ACIDOSIS.

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ABSTRACT

The development of metabolic acidosis during cardio-pulmonary bypass (CPB) is a well recognized but poorly explained phenomenon. It has been hypothesized that it is purely a development after the delivery of pump prime. A retrospective study was conducted at our hospital on 68 patients who underwent elective coronary artery bypass grafting (CABG). Sampling of arterial blood was performed at three time intervals: (T₁) Baseline, prior to induction; (T₂) 5 minutes after initiation of CPB and prior to administration of cardioplegia solution; (T₃) during rewarming prior to weaning the patient off CPB. Measurements of Na⁺, K⁺, Cl⁻, pH, pCO₂, HCO₃⁻, Base excess, Anion gap, Strong ion difference at each collection point were performed. Results were analyzed in a quantitative manner. On delivery of pump prime, all patients' developed metabolic acidosis. However, it is very important to distinguish the metabolic acidosis as their management varies. Anion gap has been found to be useful in managing peri-operative metabolic acidosis.

KEYWORDS:

Metabolic acidosis, C.A.B.G., Anion gap, Pump prime.

INTRODUCTION

Cardio-Pulmonary bypass has long been recognized as being associated with a metabolic acidosis. Several causes have been suggested to explain this phenomenon including an increase in serum lactate and chloride levels, yet the etiology of such changes have not been clearly understood:

Schengraber *et al* in 1999 demonstrated that chloride rich fluids delivered in appropriate quantities over a short period of time could induce acidosis. A similar situation can occur on initiation of C.P.B. wherein there is a rapid delivery of fluid to the patient. Delivery of pump prime fluid can amount to almost 50% of plasma volume in most patients. It is therefore possible in such a situation to induce an immediate metabolic acidosis whose characteristics would however depend upon the type of pump prime used.

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MATERIALS & METHODS

This was a retrospective study conducted at our hospital. The data of 68 patients who underwent elective C.A.B.G. during the last year was compiled. The exclusion criteria were:

- ★ Age > 70 years
- ★ Serum Creatinine > 1.3 mg/dl
- ★ Anemia (Hb < 10g/dl)
- ★ Pre- existing acid base abnormalities

All patients received 30 ml/kg of Ringers lactate solution as the priming fluid on the C.P.B. circuit. This is the standard priming solution used in our Institute. The constituents of this solution are shown in Table 1.

C.P.B. was performed using a membrane oxygenator (Univox Spiral Gold-Baxter). The pump flow rate was set at 2.4L.m². min. and the body temperature was in the range of 30-34°C. The sampling of arterial blood was performed at three time intervals: (T₁) baseline ABG prior to induction of anaesthesia; (T₂) 5 minutes after initiation of C.P.B. at full flow and prior to administration of the first dose of cardioplegic solution; (T₃) during

rewarming prior to weaning the patient off C.P.B. The A.B.G. was analyzed on an AVL-2 compact series A.B.G. analyzer and the serum electrolytes were analyzed on an AVL 9883 electrolyte analyzer. The results were then analyzed in a quantitative manner.

RESULTS

The data was collected and analyzed in the following manner. First, the median value with 95% confidence interval was calculated for each variable at the three time points: T_1 , T_2 & T_3 . The data was then tested for normality using skewness measurements. As the data was not normally distributed, Friedman's statistics for multiple comparisons of non parametric data was used to test if there was any significant change over the time $T_1 \rightarrow T_2 \rightarrow T_3$. For measured variables that showed significant change over this time frame, a Willcoxon ranked sign test, adjusted for multiple comparisons, was performed to obtain an estimated median change (97.5% confidence interval) over the following time periods: $T_1 \rightarrow T_2$, $T_2 \rightarrow T_3$ and $T_1 \rightarrow T_3$. The results are summarized in Table 2. There was no significant difference with respect to age, sex, weight, pump time, number of grafts and the surgical time in the study group.

DISCUSSION

On delivery of pump prime fluid, all patients developed a metabolic acidosis (base excess 1.01 mEq/L [T_1] to -2.03 mEq/L [T_2] {p value < 0.001}). This is very similar to the study done by Frank J. Liskaser et al in 1999 who showed that on delivery of pump prime fluid (Haemaccel-Ringers lactate solution) all patients developed a metabolic acidosis. They concluded that the mechanism of metabolic acidosis was basically hyperchloremia (1). A similar study done by Harris et al in 1970 concluded that a degree of metabolic acidosis does occur during the initial period of C.P.B. in humans (2).

It is very important to distinguish metabolic acidosis as their management varies. The anion gap is a useful, if imperfect, tool for distinguishing metabolic acidosis such as keto-acidosis, lactic acidosis and uremic acidosis, that increase the anion gap from hyperchloremic acidosis, in which there is no increase in anion gap. Scheingraber *et al* who showed the usefulness of assessing the anion gap in managing peri-operative metabolic acidosis has highlighted this fact (3,4).

The administration of Ringers Lactate solution induced a degree of dilutional hyperchloremic acidosis. The following time period (T_2 - T_3) was characterized by a corrective physiological response with a decrease in serum chloride towards baseline. Accordingly, there was a significant improvement in the degree of metabolic acidosis with an increase in base excess.

The importance of hyperchloremia is emphasized by Stewart's mathematically based approach to acid base balance (5). According to Stewart, the major determinant of hydrogen ion concentration is the strong ion difference (SID) in the body. In a solution containing any collection of strong electrolytes, the hydrogen ion concentration is determined by the difference between the sum of the sodium and potassium concentration and the chloride concentration ($SID = Na + K - Cl$). A decrease in SID is associated with a metabolic acidosis and an increase in SID is associated with a metabolic alkalosis. Change in chloride concentration is the major anionic contributor to change in hydrogen ion homeostasis. The development of significant hyperchloremia with large volumes of fluid would result in a decrease in SID and metabolic acidosis. Furthermore, the Stewart model is able to quantify the effect that electrolyte changes have on base deficit.

The concept of lactic acidosis cannot be ruled out, as the plasma lactate levels were not estimated during the peri-operative period. According to Stewart's framework one of the immediate major effects of bolus administration is dilutional hypoalbuminemia, which was not estimated in our study.

At this stage the question is whether hyperchloremic metabolic acidosis is hazardous or not? Most of the evidence suggests that it is not. However, correct treatment is absolutely dependent on differentiation of hyperchloremic acidosis from lactic acidosis. Aggressive attempts to improve organ perfusion, based on misdiagnosis of lactic acidosis, could prove harmful. This data shows the value of using the anion gap to support the diagnosis of acute dilutional hyperchloremic acidosis. This observations done by Scheingraber *et al* emphasizes the fact that in the presence of an acute decrease in serum albumin, the anion gap should be decreased proportionately; if it is not, other causes of metabolic acidosis should be considered (3).

CONCLUSION

C.P.B. associated metabolic acidosis appears to be iatrogenic in nature and derived from the effect of pump prime fluid on acid base balance.

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TABLE 1
Concentration of ions in the prime fluid

Strong Ion	mEq/L
Sodium	131
Potassium	5
Chloride	111
Calcium	4
Bicarbonate	29

Table 2
Median concentration of measured variables over three time points.

Measured Variable	Median value of measured variable		
	T ₁	T ₂	T ₃
Sodium	139	140	140
Potassium	3.8	3.8	4.3
Chloride	101	112*	104**
pH	7.337	7.327	7.434
PCO ₂	41.0	31.9*	32.4 +
HCO ₃	23.6	16.2*	23.5**
Base excess	1.01	- 2.03*	-1.01**
Anion Gap	18.2	15.6	16.8
Strong Ion difference	41.8	31.8*	36.3

* Statistically Significant ('p' value < 0.01) from T₁ to T₂

** Statistically Significant ('p' value < 0.01) from T₂ to T₃

+ Statistically Significant ('p' value < 0.01) from T₁ to T₃

Strong Ion difference (Sodium + Potassium - Chloride).

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